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ULCER OF THE STOMACH AND DUODENUM CANCER OF THE STOMACH

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The theories which are today advanced to account for the causation of peptic ulcer increase constantly in number and the different factors, emphasized by different investigators, present a bewildering array of divergent thoughts. It is difficult to distil from the whole mixture a satisfactory explanatory product for the reason that so many various things are involved. Dietary irregularities, deficiency of vitamins,[51] infection, local tissue susceptibility, central nervous system lesions,[23,58] and other abnormalities, may all play a part.[37,38] The recent use of the flexible gastroscope has made us more conscious of the frequent occurrence of chronic gastritis, thought by many to be at times the precursor of ulcer, and perhaps of cancer of the stomach.

The first outstanding theory and the most reasonable one, is that ulcer may be initiated by the failure of the normal neutralization of the acid gastric juice by the highly alkaline duodenal contents which are ordinarily allowed to flow back through the pylorus into the stomach. This is due to failure of relaxation of the pyloric sphincter, achalasia, which in turn may be caused by reflex nerve impulses upsetting the sympathetic and parasympathetic control of the pyloric muscle. It is thought that irritative processes anywhere in the abdomen, as, for instance, the gallbladder and appendix, may contribute to this condition.

Secondly, it is universally accepted that there is a definite class of high-strung, nervous people, who have an ulcer diathesis or individual tendency to the disease; this diathesis may even have a definite familial trend.[20]

Thirdly, there is the belief that there occur certain local areas of decreased resistance,[20] but as to the real cause of these we are ignorant. Certainly it is true that most gastric ulcers are on the lesser curvature where the food is rubbed along by the peristaltic waves. Practically all duodenal ulcers are within an inch of the pylorus where the acid gastric contents are forcibly squirted against the duodenal mucous membrane.

Accepting for the moment the fact that the question of etiology is not satisfactorily solved, I pass on to the consideration of ulcer of the stomach. This leads me to my first precept:

Never speak loosely of peptic ulcer, but always qualify the expression by saying duodenal or gastric ulcer, because a gastric ulcer may be a cancer, while a duodenal ulcer never is.

Gastric ulcer occurs usually on the posterior wall along the lesser curvature; and ulcer on the greater curvature is always cancer. An ulcer within one inch of the pylorus is cancer nine times out of ten.[27] It is fitting here to insert precept number two:—

Ulcer, if it is ulcer, whether gastric or duodenal, is, at the outset, a medical disease.

The one most important and never-to-be-forgotten point, however, in the diagnosis and treatment of gastric ulcer, is the decision as to whether or not it is cancer. An early cancer may be an ulcerative lesion, indistinguishable by any clinical test from benign ulcer.[11,40,47,49,50] Not many years ago we were taught that most cancers developed on preexisting ulcers, and thus an ulcer could be considered as a pre-cancerous lesion. This dictum did not long go unchallenged and has been greatly modified. A recent article on this subject demonstrated that 3.75% of the ulcer cases became malignant and 13% of definite cancers revealed evidence of previous benign ulceration.[42] Segal and Scott have reported 101 cases of gastric ulcer; in this series seven which were considered benign turned out to be malignant and eleven which were thought to be malignant turned out to be benign. Hinton says, "once an ulcer, always an ulcer," but he adds that in 7% or 8% of the cases one cannot tell

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whether one is dealing with a true benign ulcer or an ulcerating carcinoma.[26] Scott presented ten other cases in which differention between gastric ulcer and cancer was impossible. He notes that, in 100 cases of gastro-enterostomy done by Balfour for benign gastric ulcer, six later died of cancer.[49] The early symptoms of cancer may be no more than loss of appetite, sometimes with easy fatigability, pallor and weakness.[59]

The most recent valuable addition to our diagnostic instruments is the gastroscope, a very expensive apparatus which requires expert skill to use.[6, 7, 8, 43, 48] Suspicion on the part of the attending physician is the first important step in diagnosing cancer of the stomach; the suspicion should be doubled if the ulcer is a large one.

Given a case of stubborn or newly occurring gastric indigestion in a person of middle or greater age, investigate the stomach with all available dianostic material.[14, 16, 17, 29] If a diagnosis of benign ulcer is made, put the patient on a careful regime and in three weeks repeat the x-rays. If the lesion is larger, explore at once; if in doubt, explore at once; if the lesion is smaller, one may carry on the medical treatment, but watch carefully. If the patient is a laboring man who cannot carry out an adequate dietary regime, operation is preferable to a "laissez-faire" policy. To sum this up, I am led to precept number three. *"In every case of gastric ulcer, think of cancer, and, if you are in the slightest doubt, insist on operation."*[24]

I will digress here a moment to discuss the prognosis after adequate treatment, medical or surgical, of gastric ulcer and cancer. A gastric ulcer, once healed, requires less medication and is less liable to recurrence than a duodenal ulcer.[25, 32] In cancer the life expectancy after operation can be best predicated on the degree of malignancy as estimated by Broder's method; of Grades I and II, 55-63% will live five years or more. Of Grades III and IV, only 20% will live five years.[4] Better surgical results in cancer of the stomach will come not from improvement or extension of the operative technique but from earlier diagnosis by the attending physician. The solution of the problem depends on those of you gentlemen who are doing general medical practice.

Duodenal Ulcer

In considering duodenal ulcer I pass at once to the question of treatment and reiterate that this ulcer is, except in complicated cases, purely a medical disease, to be treated conservatively. This is particularly true in young patients who hardly ever do well with surgical operation. In a recent careful review Sandweiss[46] concludes that histidine (Larostidine), Synodal (emetine), and vaccines are of no special value. Larostidine has been particularly advocated but there is no sound evidence to prove that it has any great merit; Barry and Florey,[5] experimenting on animals, say it has no effect in either preventing or curing ulcers. Another writer, Bulmer[9, 10] from England, reports that it should be used merely as an adjunct to dietary treatment. Upham and Barowsky[54] ran a series of cases with histidine and a parallel control series in which sterile water was injected. The latter did just as well as the former. Every case must be treated individually—no set rules can be laid down. The use of insulin, as advocated by Sperber[52] of Providence, has given him good results, but I have not tried it nor seen other reports as to its use. Mucin, obtained from hogs' stomachs and given by mouth to form a protective coating over the stomach wall, has certainly given some good results. Nothing has, to my knowledge, proved better than a careful dietary regime according to a strict or modified Sippy method.

Operation should be reserved for cases with intractable symptoms of long duration, pyloric obstruction, hemorrhage or perforation. Furthermore, patients with high gastric acidity do not do so well with operation because of the tendency of the acid to cause subsequent ulcer formation.[3, 13, 56, 57]

The type of operation is also a matter of great importance. A few years ago we believed that in any uncomplicated duodenal ulcer a posterior gastro-enterostomy was the best procedure; today we know that in many cases it fails, either because the symptoms are for some unexplainable reason not relieved, or because a second ulcer, jejunal, gastro-jejunal, or a gastro-jejuno — colic fistula, may result. The figures as to the frequency of this secondary ulcer vary with different authors from 2% to 48%.[33, 60] For duodenal ulcer with pyloric obstruction, however, especially with low acid, simple gastro-enterostomy may be done. The opinion of the most experienced surgeons in this country is swinging around to the belief that we should do fewer surgical operations for duodenal ulcer, and when we do do them, they should be more radical. I must mention here, however, that for this ulcer, with or without obstruction, the simpler pyloro-

plasty (Judd), with or without excision of the ulcer, is gaining many adherents. I have used it with excellent results. This leads me to Precept No. 4: *"Do not operate on duodenal ulcers until you are forced to do so."*

To ensure a good result after an operation for any ulcer, one must insist that the patient continue for a long time, if not forever, on a rather careful diet. As to smoking before or after operation, men vary in their opinions, but I feel convinced that the likelihood of cure is greater if smoking is given up. I cannot produce figures to prove this, but if smoking has a harmful effect in thrombangitis-obliterans, as we know it does, it is reasonable to suppose that it may have some deleterious action on the terminal vessels in the stomach and duodenum.

Jejunal Ulcer After Gastro-enterostomy

One can lay down no set rules for the handling of a jejunal ulcer. Medical treatment should be given a fair trial, and if this fails, as it usually does, surgery becomes unavoidable. The gastro-enterostomy should be taken down and then the subsequent procedure will depend on the individual case; it is safe to say that in most instances a gastro-duodenostomy, as for instance, the Judd method, is the operation of choice.[18, 33, 36, 60]

Perforation[53]

Almost all acute perforations are in the duodenum or at the pylorus. Gastric ulcers, usually located on the posterior wall near the lesser curvature, are characterized by a slow penetration into the posterior retroperitoneal structures, without the sudden onset so characteristic of an anterior perforation and sudden flooding of the peritoneal cavity with stomach and duodenal contents. These sudden outpourings from an ulcer of the duodenum result in a drifting of the fluid down the right side, or gutter, of the abdominal cavity, with puddling around the caecum. If the leak is slow the symptoms and signs may at first be referred to the appendix and a wrong diagnosis made. Besides the diagnostic value of discovering a decrease in the area of liver dullness, an x-ray will frequently show gas between the liver and the diaphragm. This finding is pathognomonic.

The one controversial question which has to be discussed is "How much shall one do at the operation?" Recommendations vary all the way from simple closure of the perforation to radical gastric resection.[35] An excellent recent article by Graham[22] of Toronto advocates simply closing the hole by suturing some omentum into it; on the other hand, following simple closure, recurrence of symptoms is claimed by Sallick,[45] in between 64% and 71%; other authors give lower figures. Lewisohn reports persistence of symptoms in many cases but reserves his more extensive operations for a later date. From Europe has come the advocacy of extensive resection[34]; this had not been generally accepted in this country. Certainly, in the cases I have seen, I would not feel like subjecting the patients to it; after all, one should not shoot off all of one's ammunition the first time. I would, however, in a case operated on within six hours, consider gastro-enterostomy if I were convinced that there was going to be a considerable degree of obstruction at the pylorus. In any event, the surgeon must do only what he feels competent to do quickly and dexterously. The more radical procedures must be left to those with more self-confidence and more experience than most of us have. This brings me to:

Precept No. 5: *Operate on a perforated ulcer as soon as possible, and be conservative.*

Hemorrhage

As one author says, there are three difficulties in reaching the correct decision as to what is proper to do with a bleeding ulcer—1, the worry because one cannot predict whether the bleeding will or will not stop; 2, the desire to operate for persistent bleeding, even when operation may be dangerous; and 3, the question whether the bleeding really comes from an ulcer at all—it may be from an oesophageal varix or a diffuse gastritis. Reschke has recently compiled some statistics to the effect that in cases with moderate hemorrhage the mortality is 9.5%, but in severe hemorrhage it is 17-27%. Balfour[2] says that the worst cases are those which bleed severely while in the hospital being treated for ulcer, and thinks these are the only ones in which the indications for operation are clear-cut and imperative. Meulengracht advocates immediate feeding, with careful diet, in presence of hemorrhage, and in 300 cases claims a mortality of only 1%. There are certain sound reasons for this. It seems worth while trying. I have as yet had no experience with it.

Our method of handling acute hemorrhage is as follows:—Put the patient to bed, give nothing by mouth for 12-24 hours, nothing parenterally; and morphine by the clock. Begin fluids by vein or hypodermoclysis cautiously. Transfuse only if the pa-

tient's condition becomes alarming. Then wait and continue as above. If there is more bleeding, give one more transfusion. If the bleeding persists, go ahead with operation, having donors on hand for at least two more transfusions. At operation the bleeding point must be exposed and ligated, if necessary by opening the stomach or duodenum.[61, 62, 63] I now reach the last Precept, No. 6. *Do not operate on a bleeding ulcer until you think the patient is going to die if you do not operate.*

Conclusion

Balfour has written a book of 913 pages on this subject, mostly on ulcer and cancer of stomach, and ulcer of the duodenum, and I have tried to cover it in 20 minutes. The evening will prove to have been worth while if you carry away one thought, and that is this:—

"Never forget that an ulcer of the stomach may be cancer."

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THE CHEMISTRY AND MODE OF ACTION OF SULFANILAMIDE AND RELATED COMPOUNDS

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The contributions of Ehrlich to the chemotherapy of syphilis had led to the hope that bacterial infections in general would prove amenable to this type of treatment, but, with a few exceptions, the record until 1935 was one of discouraging failure. It was in that year that Domagk[1] published the results of his work on the action of a group of azo dyes on experimental streptococcal infections in mice, and started medical science on a series of researches which have done more to advance the chemotherapy of infectious diseases than had been accomplished in all the preceding years.

Domagk's most important contribution was the observation that compounds of relatively low bactericidal effect, or none at all, in vitro, may prove very effective in the animal body. He was particularly successful in overcoming hemolytic streptococcal infections in mice by the administration of the hydrochloride of sulfamidochrysoidine, or 2: 4-diaminoazobenzene-4'-sulfonamide (Figure 1) later known by the proprietary names of Streptozon and Prontosil. Even before he published his results, the therapeutic action appeared so promising that several physicians had tried this compound on some of their patients, and their favorable reports appeared in the literature before that of Domagk.

Prontosil therapy was handicapped by the low solubility of the free base and the acid reaction of the more soluble hydrochloride, both of which interfered with its parenteral use. Prontosil Soluble was developed later, and is the one now available in 2.5 percent solution. The publication of Domagk's results naturally brought other investigators into the field. Tréfouël, Tréfouël, Nitti and Bovet[2] found that most of the effective dyes contained benzenesulfonamide attached to one of the nitrogens of the diazo group. They suggested that the animal was probably able to break the dye molecule at the diazo linkage to produce the para-amino derivative of benzenesulfonamide, which is the amide of sulfanilic acid, or sulfanilamide; and they were able to show that this compound had an action similar to that of Prontosil in protecting mice. Their results were soon confirmed by Buttle[3] and his associates, who also demonstrated the lower toxicity of sufanilamide, as well as its value in meningococcal infections in mice. It was found capable of protecting mice infected with 10,000 fatal doses of streptcocci, and the protective action against meningococci was shown to be of a high order. Figure 1 shows the structural relationships of Prontosil, Prontosil Soluble and sulfanilamide.

The drug was introduced into this country as Prontylin, and is now obtainable under this and other names from several reliable American manufacturers. Among the other names found in the literature are para-aminobenzenesulfonamide, para-aminophenylsulfonamide, and sulfanilamide.

Distribution and Determination. With any of the commonly used methods of administration, sulfanilamide soon reaches all parts of the body, its distribution being governed chiefly by the water content of the tissues and fluids. Part of it is converted to para-acetylaminobenzenesulfonamide, and both are found in the urine. Inasmuch as the rate of excretion is variable, it is advisable to check the suitability of the dosage by quantitative determina-

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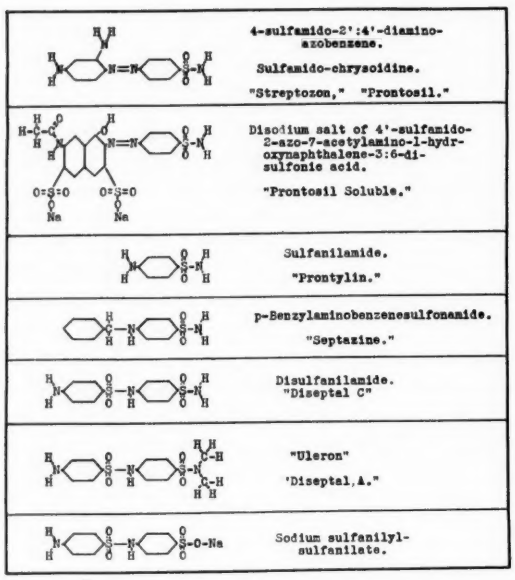


FIGURE 1

tions of the concentration in the blood, especially in patients who do not show the customary response, or who react unfavorably. Several methods suitable for such determination have been described in the literature.

Our experience has been limited to the methods of Marshall[4] and Schmidt.[5] Schmidt's is the simpler of the two, and has several advantages. It requires fewer and less expensive reagents, lessens the working time, and permits determinations on the regular tungstic acid blood filtrates.

Mode of Action. The mode of action of this group of compounds has been the subject of repeated investigations. The observation that Prontosil was active only after being introduced into the animal was one of the reasons leading to the conclusion that it was not effective per se, but was probably modified in the body to produce the real therapeutic agent. It was shown that reduction in vitro would break the molecule at the diazo bond and that one of the reduction products, sulfanilamide, was as effective as Prontosil in streptococcal infections. Colebrook, Buttle and O'Meara[6] found that sulfanilamide in culture media and in blood was definitely bacteriostatic, and bactericidal if the number of organisms was small. They also showed that the blood from persons who had taken either Prontosil or sulfanilamide was detrimental to the growth of hemolytic streptococci.

Those organisms that are resistant to the bacteriostatic action of sulfanilamide in vitro are usually equally resistant to its effect in vivo. This is well shown in a paper by Bliss and Long,[7] discussing the failure of sulfanilamide therapy in four cases of pyelitis and cystitis. The betahemolytic streptococci isolated from these patients were found to be in Group D of the Lancefield classification, and suffered no inhibition of growth in broth containing 1-10,000 sulfanilamide. Several Group A strains, on the other hand, were so susceptible to the action of the drug that the 18-hour cultures in 1-10,000 sulfanilamide developed only 1/100,000 as many organisms as did the controls. It is with beta-hemolytic streptococci of this group that most of the successful experimental and clinical work has been done.

Another paper[8] by the same authors deals with the treatment of experimental Clostridium welchii infections in mice. The first peritoneal smears from both treated animals and controls contained leucocytes loaded with organisms, and many free bacteria. Subsequent smears showed the same degree of phagocytosis, but fewer free bacteria in the treated mice and progressively increasing numbers in the controls. Six hours and fifteen minutes after infection all the control mice were dead while the treated animals were well on the way to recovery, and in twenty-four hours free bacteria were no longer present in the exudates. In a similar series of experiments using hemolytic streptococci, phagocytosis was greatly increased by treatment. Osgood and Brownlee[9] point out that in the presence of sulfanilamide streptococci are less liable to cause hemolysis of red cells, and conclude that "The major action of sulfanilamide on the beta-hemolytic streptococci seems to be the neutralization of toxin." Their experimental results do not, however, exclude the possibility that the drug inhibits toxin production.

Our attempts to understand the action of sulfanilamide are handicapped by the lack of agreement of the reports in the literature, but this confusion is not surprising if we bear in mind the fact that the effects of sulfanilamide vary qualitatively and quantitatively with species, type, and even the strain of organisms used. The effect on the host is also subject to species variation and individual idiosyncrasy. At present it seems safe to say that the more recent work has not furnished adequate reasons for discarding the earlier hypothesis that the most impor-

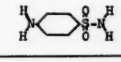
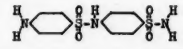
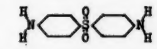
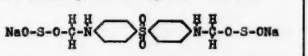
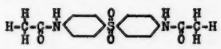
tant factor in this type of therapy is the directly damaging action on the bacteria. As a result, they increase less rapidly, become more susceptible to phagocytosis and less capable of producing toxin. The natural defense mechanisms of the host are then able to destroy the organisms faster than they are produced.

A year ago there was a growing impression that sulfanilamide was practically the last word in this type of therapy and that the more complex compounds could be discarded. We are not so sure of that now. There is evidence that in some infections the more complex compounds may be better. Rosenthal, Wooley and Bauer[10] found that two-thirds the maximum tolerated dose of Prontosil would save 60 percent of mice infected with one fatal dose of the virus of lymphocytic choriomeningitis, and 30 percent of those receiving five fatal doses. No appreciable effects were obtained with sulfanilamide, Prontosil Soluble, di-sulfanilamide or Chrysoidine-R. Dochez and Slanetz[11] in a preliminary report state that sodium sulfanilyl sulfanilate, Figure 1, was used successfully in the treatment of four ferrets experimentally infected with the virus of canine distemper. Twenty-six out of twenty-eight dogs treated clinically recovered; the other two were at an advanced stage when first seen and developed severe pulmonary infection. Eighteen cats also showed good response. Although several other virus diseases, including poliomyelitis in monkeys, have been studied, these two appear to be the only ones so far showing any definitely positive results with this type of therapy.

Diaz de Leon[12] has described the successful treatment of fifteen cases of tertian malaria with Prontosil. Kolmer[13] has reported on the properties of pyridine derivatives. The number of experiments is limited, but the effect on streptococcal infections in mice and rabbits is such as to warrant further study.

Rosenthal[14] and his associates have made an excellent study of the effects of several sulfur compounds on the pneumococcus. They showed the practical advantages of combining specific serum with chemotherapy, by adjusting the infecting dose of organisms so that less than 12 percent of the mice survived by either treatment alone. When the two were combined, 40 to 45 percent survived.

It has been shown that compounds not so closely related to sulfanilamide may be of value. As far back as 1934 Rosenthal[15] found sodium formaldehyde sulfoxylate effective in treating mice infected

	M.E.D.	M.T.D.	T.I.
Sulfanilamide 	0.75	2.5	3.3
Disulfanilamide 	1.6	8.0	5.0
Diamino diphenylsulfone 	0.025	0.15	6.0
Formaldehyde sulfoxylate sulfone 	0.2	3.0	15.0
Diacetyl diamino diphenyl sulfone 	0.2	>4.0	>20.0

M.E.D.: Minimum Effective Dose.
M.T.D.: Maximum Tolerated Dose.
T.I.: Therapeutic Index, $\frac{M.T.D.}{M.E.D.}$

FIGURE 2

with one particular strain of Type I pneumococcus, though of no value against other strains of the same type. Amino methyl sulfoxylic acid had a similar action, showing that activity did not depend on the formaldehyde group. Bauer and Rosenthal[16] have studied the action of a group of substituted diphenyl sulfones and the corresponding sulfides. Some of these may prove to be superior to sulfanilamide. Figure II shows a few of these, with the minimum effective dose, maximum tolerated dose, and therapeutic index for each.

Summary. At present, the most important of the new chemotherapeutic agents is probably sulfanilamide. It has been found highly effective against several kinds of bacteria, especially the meningococcus and the more virulent forms of beta-hemolytic streptococcus. Other micro-organisms show varying degrees of susceptibility, and some are very resistant. Several other compounds, some of them closely related to sulfanilamide, have been found more effective against pneumococci, malaria and some virus diseases. A few of the newer ones show a lower relative toxicity for experimental animals, and therefore have the advantage of a more favorable therapeutic index, and permit the use of a higher dosage. The value of the most of these has not as yet been fully established by adequate clinical experience. Most of the compounds so far proven

valuable consist of one or more benzene rings to which are attached nitrogen and sulfur containing radicals, but the derivatives of pyridine apparently deserve further investigation. The toxicity is not the same for all species of animals; these drugs must therefore, even after the most careful animal experimentation, be used cautiously on human subjects.

The evidence available in the literature indicates that the therapeutic effect of sulfanilamide is probably due in large part to a directly damaging action on the organisms, resulting in bacteriostasis, greater susceptibility to phagocytosis, and impaired ability to produce toxin. The host is then able to destroy the bacteria faster than they are produced.

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Rhode Island Hospital

SCHEDULE FOR SEPTEMBER, 1938

MONDAYS:

September 5, Labor Day
I Surg. Grand Rounds September 12, 26
II Surg. Grand Rounds September 19
Surgical Grand Rounds 10:00 A. M.
Thoracic Clinic 4:30 P. M.

TUESDAYS:

Gastro-Intestinal Clinic 9:30 A. M.
Surgical Grand Rounds 10:00 A. M.
I Surg. Grand Rounds September 6, 20
II Surg. Grand Rounds September 13, 27

WEDNESDAYS:

Tumor Clinic 10:00 A. M.

THURSDAYS:

Orthopedic Grand Rounds 9:00 A. M.
Thoracic Clinic 11:30 A. M.
Gyn. Staff Meeting 8:30 P. M. September 1

FRIDAYS:

INTERNS REUNION SEPTEMBER 9, 10
Fracture Grand Rounds 11:00 A. M.
Pediatric Grand Rounds September 2, 16, 30
G. U. Staff Meeting 7:30 P. M. September 2
Surg. Staff Meeting 8:30 P. M. September 2

SATURDAYS:

Neurological Grand Rounds 9:00 A. M.
Medical Conference 10:00 A. M.

Memorial Hospital

A new active dental service has been established at the Memorial Hospital. The main purpose of the service will be to find, and eliminate where necessary, all foci of infection having a direct bearing on the rapidity of recovery of hospitalized patients. The dental staff is anxious to give and receive the utmost cooperation in the interests of the patients in the Hospital.

The following four doctors have entered The Memorial Hospital for Internship of 18 months: Kenneth Cedric VonPohle, M.D., Los Angeles, California. Duncan Hector Campbell Ferguson, M.D., Pawtucket, R. I. William Joseph O'Connell, M.D., East Providence, R. I. Howard William Umstead, M.D., Providence, R. I.

Two will arrive September first: Roy Walter Nelson, M.D., Attleboro, Mass. Bert Simmons Jeremiah, M.D., Providence, R. I.

Mr. Adrien G. Tetreault of Central Falls has completed two months as a medical student at The Memorial Hospital.

ANSWERS TO COMMON QUESTIONS

CHARLES BRADLEY, M.D.
EAST PROVIDENCE, RHODE ISLAND

It is essential that parents understand as clearly as possible the nature and treatment of so dreaded and vague a syndrome as convulsive disorders. In practice certain questions arise with unusual frequency. It may be helpful to anticipate some of them for which reason the following common requests for information are given specific answers.

"Has my child epilepsy, doctor?" There appears to be no advantage in using the term epilepsy at the present time inasmuch as it implies a discouraging prognosis and is associated with a certain amount of social stigma. It should be explained to the parents that the convulsion is a symptom of some underlying disorder and that every attempt must be made to ascertain its cause. Even in the absence of removing the etiology, treatment may be effective, and except in long-standing disorders where mental deterioration has already taken place, a discouraging prognosis is not warranted unless adequate treatment fails.

"Are convulsions often due to worms?" Intestinal parasites were formerly blamed for a great variety of pediatric disorders, the number of which has gradually diminished with the spread of medical knowledge. In the light of our present knowledge, convulsions are probably very rarely caused by worms. If there is definite indication that worms are present, they should of course be treated.

"Will my child outgrow his fits?" A very few children who have repeated convulsions recover spontaneously without treatment. There is no accurate way of knowing in advance which children will have this fortunate outcome and in no case should it be awaited with any degree of confidence.

"What can my child eat?" Unless the child is being definitely treated by one of the accepted forms of dietary therapy, only the ordinary commonsense restrictions of his diet are necessary. Over-eating, constipation, etc., undoubtedly precipitate occasional seizures in susceptible children, but ordinary rules of hygiene do not permit of excesses.

"Is epilepsy inherited?" Granted that the term epilepsy serves no very good use in clinical practice, it must be stated that the direct inheritance of most nervous and mental disorders is still a matter of some conjecture. Unless there is a definitely large incidence of convulsive disorders in the immediate family or antecedents, the child's convulsions are probably not caused by any defect or metabolic disturbance which has an hereditary basis.

"Is there any hope for the child who has convulsions?" The investigation of these conditions should always be carried out in a hopeful spirit, and no definite prognosis should be given until the search for a cause has been exhaustive, and various forms of therapy have been tried. A working hypothesis that one-third of all patients can be completely relieved, one-third aided, and one-third not helped at all, regardless of the etiology, is one practical outlook on this situation. Most treatment is still empirical but fortunately fairly successful.

"Should I allow my child to get tired?" For most children with convulsions, particularly if their parents are solicitous enough to ask this question, a fairly busy routine should be planned. A happy life of activity leaves little time to anticipate seizures and is some assurance that they will not recur as frequently as expected. The author has seen very few children in whom limitation of activity has decreased the number of seizures.

"Will injections help my child?" Although many useful drugs are now available in hypodermic form, all of the reliable methods of treatment of convulsive disorders may be given by mouth except in emergencies.

"Will it harm other children to see my child having a fit?" The attitude of children towards such affairs is almost entirely determined by the attitude of the adults who are with them. A calm, matter-of-fact attitude in dealing with a child who is having a convulsion will not upset other children, whereas consternation, excitment, and pity will certainly disturb most children.

"Can fright cause convulsions?" Any emotional upset may precipitate an individual seizure in a patient who is susceptible to convulsions. There is no very definite evidence that emotional upsets in themselves are the primary causes of seizures.

Continuation of a paper on "Practical Management of Convulsive Disorders in Childhood." 21: 2, 27, Feb. 1938.

Memorial Hospital

Entrance to MacColl Building

Reprinted by courtesy of the Superintendent of Memorial Hospital

THE RHODE ISLAND MEDICAL JOURNAL
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JAMES R. MacCOLL

James Roberton MacColl was born in Glasgow, Scotland, April 2, 1856. He died at his home in Providence on November 23, 1931, in his seventy-sixth year. His father, Hugh MacColl of Glasgow, was a descendant of the MacColls of the Island of Mull, visited by Samuel Johnson and James Boswell on their "Tour of the Hebrides" in 1773. The mother, Janet Roberton MacColl, was the daughter of a prosperous iron founder of Glasgow. James R. MacColl took a course at Glasgow Technical College and early in life achieved success and reputation in Glasgow as a manufacturer of dress goods. In 1882, he came to Pawtucket, Rhode Island, to take the position of manager of the Lorraine Mills.

Mr. MacColl soon become a prominent figure, not only in the textile industry but also in varied civic and national affairs. He was a member of the Tariff Committee of the National Association of Wool Manufacturers, a director of the Chamber of Commerce of the United States from 1914 to 1920, a member of the American Committee of the International Chamber of Commerce, Regional Vice-President of the American Arbitration Association of 1929. He also found time to engage in charitable work. He was President of the Rhode Island Anti-Tuberculosis Association from 1908 to 1910 and a Vice-President of the Providence Community Fund, Inc. in 1930 and 1931. He was one of the incorporators and a director of the Pawtucket Memorial Hospital. Beside his financial contributions to that institution his personal interest was constantly evident in his work as a member of the Board of Directors up to the time of his death.

Mr. MacColl was married in 1884 to Miss Agnes Bogle of Glasgow. They had five sons, who survive. The only daughter, Margaret MacColl, died in 1893 at the age of five and one-half years.

In 1929, the Trustees of the Memorial Hospital announced the offer of a gift from a friend who at that time requested that the name be withheld, of $200,000, for a new building for the treatment of children's diseases and for maternity cases. On June 1, 1931, the MacColl Building was opened for patients. A bronze tablet in the reception room at the entrance states: "The MacColl Memorial, A. D. 1931. Given by James R. and Agnes B. MacColl, for Treatment of Children's Diseases and Care in Maternity, as a Memorial to their Daughter, Margaret, July 24, 1888-December 26, 1893." The architect was fortunate in that he planned a building of the greatest utility for its purpose combined with a design in perfect harmony with the graceful lines of the older buildings in this hospital group. The first floor is given up to attractive wards and rooms for the sick children and rooms for private patients. The conveniently arranged maternity department occupies the entire second floor.

We must gratefully remember the single visit following the completion of the building, only a short time before his death, which Mr. MacColl was able to make. That one time, he himself beheld the excellence of his plan.

Patience and delicacy should characterize all the acts of a physician. The confidences concerning individual or domestic life entrusted by a patient to a physician and the defects of disposition or flaws of character observed in patients during medical attendance should be held as a trust and should never be revealed except when imperatively required by the laws of the State. There are occasions, however, when a physician must determine whether or not his duty to society requires him to take definite action to protect a healthy individual from becoming infected, because the physician has knowledge, obtained through the confidences entrusted to him as a physician, of a communicable disease to which the healthy individual is about to be exposed. In such a case, the physician should act as he would desire another to act toward one of his own family under like circumstances. Before he determines his course, the physician should know the civil law of his commonwealth concerning privileged communications.

From the Code of Ethics of the A. M. A.

STATEMENT OF INVESTED FUNDS

J. W. C. Ely Fund

January 1, 1937		January 1, 1938	
37 shares Rhode Island Public Service Co.	$1,071.67	37 shares Rhode Island Public Service Co.	$1,071.67
Interest	74.00	11⅛ new shares Common Stock, Mechanics Nat. Bank	280.00
11⅛ new shares Common Stock, Mechanics Nat. Bank	280.00	Paid Rhode Island Medical Society for Journals	74.00
Interest in default	.....		
	$1,425.67		$1,425.67

Endowment Fund

January 1, 1937		January 1, 1938	
Jan. 28 Sold Oklahoma Gas & Electric Co.	$2,060.00	16 shares National Bank of Commerce & Trust Co.	$1,200.00
Coupons	50.00	74 shares Providence Gas Co.	906.50
	$2,110.00	Peoples Savings Bank	3,245.92
Jan. 28 Purchased 16 shares National Bank of Commerce & Trust Co.	$1,200.00		
74 shares Providence Gas Co.	906.50		
Postage, insurance, etc.	3.50		
	$2,110.00		
16 shares National Bank of Commerce & Trust Co.	$1,200.00		
Interest	48.00		
74 shares Providence Gas Co.	906.50		
Interest	44.40		
Peoples Savings Bank	3,076.14		
Bank interest	77.38		
	$5,352.42		$5,352.42

Frank L. Day Fund

January 1, 1937		January 1, 1938	
3,000 Canadian National Railway Co. 4%	$2,979.75	3,000 Canadian National Railway Co.	$2,979.75
Interest	135.00	Paid for Medical Books	98.43
Industrial Trust Company	411.91	Industrial Trust Company	448.48
	$3,526.66		$3,526.66

E. M. Harris Fund

January 1, 1937		January 1, 1938	
Jan. 23 Sold Central Arizona Light & Power Co.	$1,050.00	2,000 A-NY & B-NY Realizing Corp. Debentures	$2,000.00
Interest	7.08	4 shares stock A-NY & B-NY Realizing Corp.	.....
	$1,057.08	2,000 General Public Utilities	1,980.00
Jan. 23 Purchased 26 shares Nicholson File Co.	$1,040.00	26 shares Nicholson File Co.	1,040.00
Balance from sale	17.08	Paid R. I. Medical Society for Repairs on Building	305.80
	$1,057.08		

2,000 A-NY & B-NY Realizing Corp. Debentures 5½%	$2,000.00		
4 shares stock A-NY & B-NY Realizing Corp.			
Payment on prin. of above debentures and interest	91.00		
2,000 General Public Utilities Co. 6½%	1,980.00		
Interest	156.00		
26 shares Nicholson File Co.	1,040.00		
Interest	33.80		
Central Arizona Light & Power Jan. interest	25.00		
	$5,325.80		$5,325.80

Herbert Terry Fund

January 1, 1937		January 1, 1938	
96 shares Providence Gas Co.	$1,152.00	96 shares Providence Gas Co.	$1,152.00
Interest	76.80	Paid Rhode Island Medical Society for Journals	33.50
Balance on hand	528.20	Balance on hand	571.50
	$1,757.00		$1,757.00

James R. Morgan Fund

January 1, 1937		January 1, 1938	
Jan. 28 Sold Missouri Light & Power Co.	$ 515.00	43 shares Providence Gas Co.	$ 526.75
Coupon	11.25	Paid Rhode Island Medical Society for Expenses	25.80
	$ 526.25		
Jan. 28 Purchased 43 shares Providence Gas Co.	$ 526.75		
Interest	25.80		
	$ 552.55		$ 552.55

James H. Davenport Fund

January 1, 1937		January 1, 1938	
89 shares Providence Gas Co.	$1,068.00	89 shares Providence Gas Co.	$1,068.00
Interest	71.20	Balance on hand	451.93
Balance on hand	380.73		
	$1,519.93		$1,519.93

Cataloguing Fund

January 1, 1937		January 1, 1938	
Peoples Savings Bank, Clinical Conference Fund	$ 135.92	Paid out during 1937	$ 12.50
Interest	3.40	Peoples Savings Bank	139.32
Providence National Bank, Checking Account inc. Gift from Fiske Fund Trustees	220.57	Providence National Bank, Checking Account	208.07
	$ 359.89		$ 359.89

Participation Account

January 1, 1937		January 1, 1938	
Providence Institution for Savings	$ 553.47	Providence Institution for Savings	$ 567.38
Interest	13.91		
	$ 567.38		$ 567.38

RHODE ISLAND MEDICAL SOCIETY

Report of Delegate to the American Medical Association

The recent meeting of the American Medical Association in San Francisco was noteworthy in many respects. There is a marked desire of the general membership of the medical profession for self improvement in the newer developments of medical knowledge and progress of the medical societies throughout the country reflect this interest. There is also an awakened interest in the present social and economic questions confronting the country as a whole and particularly those connected with medical practice. In every part of the country various phases of this problem are being discussed. This is shown by the fact that during the past year the membership of the American Medical Association has been increased by 4,000, to the all time high of 109,435 members. This is ample evidence in refutation of the press reports of dissension in our organization. There is an awakening of physicians throughout the country to the obligations of medicine and medical practice. Social and economic conditions are inseparably intermingled with health and sickness and it is reasonable to believe that until the economic conditions are improved no satisfactory measures can be taken to socialize the practice of medicine.

The House of Delegates held four busy sessions which were attended by your Delegate. One of the most interesting resolutions was one concerning a traveling hall of health, in which exhibits of all kinds pertaining to public health and the practice of medicine may be displayed in all parts of the country under co-sponsorship with the constituent State Associations.

Much thought is being given to the status of laboratories, X-ray and physical therapy departments and the department of anesthesia in Hospitals throughout the country. With the rise of insurance plans by which insurance payments are made which guarantee medical care in hospitals to those participating in the plan, it is necessary that the above mentioned departments should not be included in these plans. Arrangements should be made for separate payments to physicians in charge of these departments, or otherwise we shall have the anomalous position of the hospitals engaging in the practice of medicine.

Perhaps the high light of the Session was the address prepared by Miss Josephine Roche, Chairman of the Interdepartmental Committee to coordinate Health and Welfare Activities of the Federal Government, which was read by Assistant Surgeon General of the United States Public Health Service, Dr. Warren F. Draper, himself a member of the House of Delegates. After hearing this address it was plainly evident that the Federal Government had undoubtedly formulated a plan in its own mind and this was borne out by the fact that at the National Health Conference, held in Washington July 18, 1938, a plan was submitted recommending the appropriation of large sums of money for the medical care of the indigent. The Health Survey of the Federal Committee is at variance with data collected by the American Medical Association and now in process of being analyzed and prepared for submission to the medical profession as a whole. It is noteworthy that the address of Miss Roche is the first communication ever received by the organized medical profession from the Federal Government. The A. M. A. in reply stated that, as always, it was ready to aid in every possible way in the establishment of a workable plan for medical care of the indigent.

The election of officers was held on the last day of the Session and Dr. Rock Sleyster was chosen President-elect. Places of meetings were selected three years in advance, in order to assure adequate hotel accommodations at reasonable rates. St. Louis was chosen the meeting place for 1939, New York for 1940, and Cleveland for 1941.

Respectfully submitted,

ROLAND HAMMOND, M.D.

AMERICAN BOARD OF INTERNAL MEDICINE, Inc.

Written examinations for certification by the American Board of Internal Medicine will be held in various parts of the United States on Monday, October 17, 1938, and on Monday, February 20, 1939.

Formal application must be received by the Secretary before September *15*, 1938 for the October, 1938 examination, and on or before January *1* for the February, 1939 examination.

Application forms may be obtained from William S. Middleton, M.D., Secretary-Treasurer, 1301 University Avenue, Madison, Wisconsin, U. S. A.